CLAIMS

What is claimed is:

1. A method for inhibiting or reducing tumor cell proliferation in an individual *in vivo* comprising:

contacting a tumor cell *in vivo* with a Rad51 inhibitor, and a polynucleotide capable of expressing functional p53 protein.

2. The method according to Claim 1 further comprising:

exposing said tumor cell *in vivo* to radiation or chemo therapies.

3. The method according to Claim 1 or 2, wherein said Rad51 inhibitor is selected from the group consisting of Rad51 antisense molecules, small molecules, peptides or antibodies.

4. The method according to Claim 1 or 2, wherein said Rad51 inhibitor is a Rad51 antisense molecule.

5. The method according to Claim 4, wherein the step of contacting said antisense molecule further comprises:

introducing to said tumor cell *in vivo* an expression vector comprising a eukaryotic functional promoter and a polynucleotide sequence encoding a Rad51 antisense molecule, wherein said polynucleotide sequence is under transcriptional control of said eukaryotic functional promoter.

6. The method according to Claim 5, wherein said expression vector is an adenoviral or retroviral expression vector.

7. The method according to Claim 4, wherein said antisense molecule is introduced locally to said tumor cell.

8. The method according to Claim 4 further comprising introducing to said tumor cell *in vivo* an expression vector comprising:

(i) a first polynucleotide sequence encoding a Rad51 antisense molecule; and

(ii) a second polynucleotide sequence encoding said functional p53 protein,

wherein said first and second polynucleotides are operably linked to one or more promoter sequences which are functional in said tumor cell to produce said Rad51 antisense molecule and said functional p53 protein

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The method according to Claim 4, wherein said Rad51 antisense molecule is selected from the group consisting of AS4, AS5, AS6, AS7, AS8 and AS9.

10. The method according to Claim 1 or 2, wherein said Rad51 inhibitor is a small molecule.

11. The method according to Claim 10, wherein said small molecule is introduced locally to said tumor cell.

12. The method according to Claim 10, wherein said small molecule is selected from the group consisting of nucleotide diphosphate, a nucleotide analogue, a DNA minor groove binding drug, a xanthine, a xanthine derivative, and halogenated pyrimidines.

13. The method according to Claim 10, wherein said inhibitor is a nucleotide analogue selected from the group consisting of a nucleotide diphosphate complexed with aluminum fluoride and a non-hydrolyzable nucleotide.

14. The method according to Claim 13, wherein said nucleotide diphosphate complexed with aluminum fluoride is selected from the group consisting of ADP.AlF4, GDP.AlF4, CDP.AlF4, UDP.AlF4 and TDP.AlF4.

15. The method according to Claim 14, wherein said non-hydrolyzable nucleotide is selected from the group consisting of ATPγS, GTPγS, UTPγS, CTPγS, TTPγS,

ADPγS, GDPγS, UDPγS, CDPγS, TDPγS, AMPγS, GMPγS, UMPγS, CMPγS, TMPγS, ATP-PNP, GTP-PNP, UTP-PNP, CTP-PNP, TTP-PNP, ADP-PNP, GDP-PNP, UDP-PNP, CDP-PNP, TDP-PNP, AMP-PNP, GMP-PNP, UMP-PNP, CMP-PNP, TMP-PNP, and hologenated pyrimidines.

16. The method according to Claim 1 or 2, wherein said Rad51 inhibitor is a peptide.

17. The method according to Claim 16, wherein said peptide is a p53 peptide having a higher affinity for Rad51 binding the p53 protein.

18. A method for sensitizing tumor cells *in vivo* to radiation comprising:

(a) introducing to a tumor cell *in vivo* a Rad51 inhibitor; and

(b) introducing to said tumor cell *in vivo* wild-type p53 protein.